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MALIGNANT HYPERTENSION AND ITS MEDICAL MANAGEMENT

by

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Introductory.

In every context, when the word "malignant" is used by our profession, in describing a disease or a disease process, there has been a tacit acceptance and an implicit confession of inadequacy of our service to the sick public. We mean to suggest that the disease is nearly always fatal and we know nothing as to why it is so. No disease is unwilling to be cured. Only, we have not gained the secret of its natural history sufficiently, nor acquired the necessary tools to deal with it effectively, and save life. It is in this sense that the term "malignant" is applied to hypertension.

Natural history of the disease.

I have reviewed the facts relative to rise of blood pressure in the normal and the abnormal way, elsewhere. It is my opinion that in the etiology of hypertension, there is a fundamental peculiarity in the smooth muscle of the arteriolar wall of individuals who develop hypertension, a hereditary or congenital anomaly, the manifestation of which is a reactivity greater than "normal" to the action of stimuli which in themselves are normal. These hyper-reactors end up as our patients on account of disturbances of structure and function in the various organs eg., the brain, the retina, the heart, abdominal viscera and the kidney. Yet, only a few of such hypertensives end up as "malignant hypertensives".

Pathogenesis of Malignant Phase of hypertension.

Blood pressure keeps rising from the time of birth until it becomes steady in the adult, except in a few, in whom it continues to rise and develop into a condition known as essential hypertension. Many have raised blood-pressure with or without symptoms, yet few turn into the "malignant stage".

What determines this pernicious turn? These unfortunates are usually young hypertensives, who have their kidney vessels damaged by a change called, arteriolar necrosis. This change is found only in the arterioles of the size of the

afferent artery to the glomerulus, the retinal arteries, etc. Byrom & Dodson as a result of experiments believe, that brief over-distension and stretching of the walls causes necrosis and lysis of the arterial muscle with subsequent seepage into the damaged muscle of plasma and blood and a fibrinoid transformation. There is adequate evidence that the renal arteriolar necrosis and cellular intimal thickening seen in the malignant phase of essential hypertension are the consequences of raised diastolic pressure (Fishberg).

This type of change and the consequent ischaemia of the kidney, releases certain amounts of vaso-constrictor material and a further rise in blood pressure. A higher diastolic pressure brings about a more diffused and rapid arteriolar and glomerular necrosis and more pressure-raising substance. A vicious cycle is thus initiated, which ends in death.

Does mere rise in pressure damage the kidneys ? Do not essential hypertensives have kidneys that are defective and affected by ischaemia ? In answer, certain facts are to be noted, which may be considered as fundamental to the production of the malignant phase. Even as no peptic ulcer is seenin the achlorhydric, so, without kidney damage, the clinical state of malignant hypertension has not been known to develop. In experimental and clinical observation the fact is noted that the higher the level at which the diastolic pressure is sustained, the greater is the percentage of malignant change. This bad change takes place less often in those who have previously diseased arterial walls such as fibrosis (arteriolosclerosis.) Malignant hypertension is never seen in the very aged, say over 70 years of age, It is with nearly-healthy vascular wall of the younger age group that this bad turn shows a maximum incidence. Arteriolar necrosis of the afferent arteriole of the kidney, the essential lesion of malignant hypertension, can occur only when there is muscular spasm followed by inadequate nourishment of the arteriolar wall. An important feature is the tempo of such change. A slow evolution of ischaemia of the kidney does not result in a malignant turn. It results only in a hypertensive or arteriosclerotic kidney. While, when the tempo is rapid, wide spread fibrinoid necrosis of the arterioles (not only of the kidney) all over the body (brain, retina, cardiac muscle, the intestines other abdominal organs) takes place resulting a variety of clinical symptoms of disordered structure and function. These are the different clinical terminations of this hypertension. There may be heart failure from hypertrophy and myocarditis, coronary insufficiency, arrhythmias etc. Cerebral apoplexy, either from haemorrhage or thrombosis may close the picture.

The changes in the kidney alone are particularly abhorrent and malignant, in that they initiate the vicious cycle referred to above. The kidney it is which is the perpetrator of this dastardly attack on the patient in a remorseless manner.

What determines which of the hypertensives will have their kidneys affected in this malignant manner ? Are there indications which foretell a malignant change possiblity ? If we knew, we might take prophylactic measures sufficiently early. Most unfortunately, the answer, is, none. We do not know in whom this change will occur. The little we know in this direction is, that the younger the patient and higher the diastolic pressure the greater is the liability to the gravamen.

Individual patients vary greatly in the height of diastolic pressure that they withstand before developing arteriolar necrosis that is the hall-mark of the malignant phase. It may be, that in the case of a youthful hypertensive, the greater elasticity of the aorta favours a higher diastolic pressure and thus the development of the malignant phase.

The clinical basis for the diagnosis of the malignant phase.

These may, by rule of thumb standards, be described as (1) a persistent unmitigated and non-fluctuating diastolic pressure of over 130 mm. of Hg. (2) retinal changes of grade III and IV, these being, papilloedema, soft exudates, haemorrhages, pallor, narrowed arteries with irregularity in size and course; (3) kidney failure, instanced by haematuria, gross and microscopic, albuminuria and N. P. N. retention in the blood.

Can the malignant phase be arrested and reversed ? What is the stage at which the malignant phase can be reversed? Cardiac failure, encephalopathy, retinal changes earlier than grade III, fair kidney eliminatory function, and a blood pressure that is responsive, are reversible. Non-reversible stage is one with gross papilloedema, kidney failure with a blood urea of over 60 mgm. per cent and which keeps rising. Anaemia and infection are the last straws.

Management of a case

As in other malignant diseases, success lies in early detection at even the very inception of the phase of malignancy and prompt and thorough treatment. As we cannot say which of the essential hypertensives will go for the bad turn, hope lies in (1) anticipation, (2) early detection and (3) arrest in the reversible stage, in that order. Progeny of known hypertensive parents from the 20th year onwards should be under constant observation. Similarly, all adults of over 35 who are hypertensives. Also, all young nephritics. Early signs are steadily rising diastolic pressure, not arrested by rest etc., albuminuria and RBC in the urine, retinal changes with tendency for blurring of the margins, retinopathy tending to grade III. Arrest of malignant phase that has come into being is difficult and good can be hoped for only by continuous and presistent attempts at therapy for periods over 3 to 6 months.

The following principles guide treatment without surgery. Each patient should be studied and the application of the principles individualised. Response or resistance to treatment, the presence of complicating factors, drug resistence, and side reactions, will guide one in the adoption of a particular drug or method. It is regrettable that many of us are guided by the readings of the baumanometer taken at random. There is no standard blood pressure reading adjudicating a patient as a hypertensive or not. " The question to be answered is, 'Is there a generalised vaso-spasm?' The only simple way of answering that question is by determining the diastolic pressure and we consider as the upper limit of normal, the diastolic pressure of 90 mm. of Hg. regardless of age " (Schroder)

Hospitalisation of the patient or near to that state, is essential till routine treatment is established for the particular patient and he is showing improvement.

The following minimum examinations should be carried out in all cases before treatment is started.

1. Weight of the patient (weekly)
2. Resting pulse, respiration.
3. Record of the urine output.
4. Blood pressure always to be recorded in the erect posture and taken at 4 hourly intervals and charted until there are few variations in it.
5. X-ray of the heart and ECG if there are indications for it.
6. Examination of the urine for albumin, RBC (microscopic) infection, (culture), pus cells casts, reaction.

7. Blood : RBC and Hb. estimation, blood urea. This must be done weekly or more often in bad cases.
8. Examination of the eye after midriasis. Note the size of the anterior chamber ; it is of diagnostic value.
9. C. S. P. pressure and chemistry in all cases having cerebral manifestations.
10. Tests for the elimination of surgical conditions of the kidney, eg., pyelitis, stone, pheochromas, urinary obstructions, etc.

The object of dieting should be to maintain the correct weight for the height and age, a little less than more. Diet must be complete in vitamin content. Protein intake should be limited to 40 to 60 gm. and calories made up with sugar and fat. Dried fruits, fresh vegetables, fruits, etc., should give bulk to overcome a tendency to constipation. Salt restriction should be enforced to the limit of compliance.

Trends in drug treatment

Reserpine is a tranquilizer and on that effect is able to reduce the blood pressure. It reduces the pulse rate. Its action in psychotic states is believed to be due to the liberation of serotonin. It is the first drug to be used in hypertension and serves as a basis for all other adjuncts. It also enhances and prolongs the effects of other hypotensive drugs. It lessens the incidence of serve reactions such as orthostatic collapse and other side effects of ganglion-blocking agents.

Hydrazinephthalazine (hydralazine, apresoline) is claimed to inactivate the pressure substances of hormonal nature produced by the kidney, and despite the fall in pressure, there is increased renal flow.

Ganglion-blocking agents act in fits and starts and do not control the blood pressure steadily. Combined with hydralazine, hyphex, Schroder claims a significant reduction in mortality from this disease.

There is danger in sudden cessation of treatment, comparable to that during treatment in juvenile diabetics. Patients' cooperation is very essential.

The following is the regimen adopted by me, regarding the use of drugs.

1. Serpasil (0.25 mgm.) t. d. s.

2. Ansolysen oral 5 mgm. 1st day ; 5 mgm. b.d. on the 2nd day ; 5 mgm. t d s on the 3rd day up to 40 mgm. t.d.s. If response is minimal it is given 4th hourly. Or it is given by injection, subcutaneously. ½ cc 1st day (2·5 mgm) ; 1 cc 2nd day. Blood pressure is taken, after ¼, ½, and 1 hour, If the blood pressure rises again, ansolysen is repeated twice or thrice a day. The object is to maintain a continuous satisfactory pressure.

3. Apresoline is added where there is no steady control over the blood pressure by the above methods. or for other reasons. It is given in small doses to start with, 5 to 10 mgm. It is better given separately than combined with serpasil in the form of adelphane.

Watch the blood pressure on a chart. Maintain diastolic pressure below 100 and the systolic below 170–190.

Do not change the drugs or stop them for the sake of a few bearable side effects. If response is present urge the patient to put up with mild side effects.

A combination of drugs gives better results and with lower dosage and is recommended.

Treat heart failure when present, with digitalis in the usual way, and the anemia with haematinics.

Continue treatment till the danger signs of malignancy disappear. There after the patient should be treated as a case of essential hypertension, but with more strict and severe control and watchfulness.

Dr. R. V. RAJAM, M.B., M.S., F.R.C.P., F.R.S.

It is with great pride that we record the election of Dr. R. V. Rajam, a revered Professor of the premier Medical College of South India, as a **Fellow of the Royal Society of Edinburgh** on the 3rd March 1958. To satisfy the curiosity of our readers, a little may be said about the Royal Society so that they might appreciate the honour bestowed upon him. The Royal Society of Edinburgh has been in existence for over 175 years. The Duke of Buccleuch was the first President of the Society. Among the many Honourable Fellows of the Society were Sir Walter Scott, Kelvin and Benjamin Franklin to mention a few of them.

To eulogise the recipient of this Honour is a colossal task. In brief his college career has been one of continued and brilliant success. A Johnstone medalist, a Master of Surgery, a Member first and later a Fellow of the Royal College of Physicians of Edinburgh, now he is a Fellow of the Royal Society of Edinburgh.

Rising step by step in the Madras Medical Service, he reached the topmost rung of the ladder when he became the Professor of Venereal Diseases and Director of the Institute of Venereology. He was for some time Principal of Madras Medical College and was the first Dean of the Madras Medical College and Government General Hospital, Madras.

The hundreds of students who have had the privilege of having been his pupils will bear testimony to the excellence of his teaching; the large number of medical

men and women who have had the opportunity to serve under him, will not fail to register the sense of discipline which he has instilled into them.

As a Research Worker, his contributions to the subject of Venereal Diseases are worldwide known. No less than 100 papers have been published by him. He brought credit to our country when he represented India at the several sessions of the World Health Organisation both in the West and in the Far East.

Besides his civilian activities, there are records to testify the useful part that he had played as a medical man in military life. He was honoured when appointed an Honorary Surgeon to the first Indian Governor-General of the country, Sri C. Rajagopalachari.

The decline of venereal diseases and what appears to be a successful control of Treponematosis in our country should be credited to Professor R. V. Rajam who has toiled day in and day out to help remove this scourge from our midst.

It is a happy augury that the useful part played by the Professor has become known far and wide and the Fellowship of the Royal Society has been conferred on him.

We wish him greater and greater Honours and hope that he will serve the State and the country more and more usefully for many a year to come.

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TREATMENT OF SCIATICA

by

Prof. Dr. SOPHUS VON ROSEN,

Professor of Orthopaedics, University of Lund and Surgeon in Chief, Orthopaedic Department, Malmo General Hospital, Sweden.

During the last 20–25 years lumbago-sciatica has become practically the most dominating form of back disease in orthopaedical practice. The reason is that it is now known that sciatica is caused by a disc prolapse with pressure on adjoining nerve roots and that the disease can be given surgical orthopaedical treatment. Before this was made clear the disease was only treated symptomatically. Anaesthetics were prescribed for pain, usually together with bed rest. If the malposition of the back was observed, and this as is well known often happens owing to the pain, orthopaedic experts talked about a sciatica scoliosis. The attempt was made to straighten this by means of different reduction manipulations, possibly combined with a plaster corset in the corrected position.

Opinions on the cause of sciatica pain were very vague and diverse. Usually it was thought that some form of neuritis was present in the sciatic nerve.

One reason that the cause of sciatica remained unknown for so long is that the disease is indeed very troublesome to the patient as long as the pain is at its height but that the pain dies away gradually and normally the patient recovers almost completely.

As far as the cause of sciatica is concerned moreover there are still several black spots to be clarified We know that sciatic pain is due to disc protrusion or disc prolapse with consequent pressure on a nerve root but why the disc occasionally splits so that the nucleus pulposus bursts through like a rupture is still unknown to us. In Sweden, Friberg and Hirsch have made comprehensive pathologic-anatomic studies showing that a disc rupture pre-supposes disc degeneration but on the other hand disc degeneration without sciatica is also very common. We know little therefore of what in the one case leads to disc rupture and sciatica and in the other case has no such effect.

It has been thought that the cause of a disc rupture is an unsuitable working position with a straight lumbar spine. The straightened lordosis would possibly force the nucleus pulposus backwards in the disc. Experience has shown that people with straight lordosis get sciatica relatively often. Heavy manual labour also seems to be a possible cause favouring the development of disc rupture.

The origin of lumbago pain is also an involved problem. It is known that the majority of sciatica patients very often have attacks of lumbago without radical pain lower down in the one leg or the other. Investigations made by another Sweden Radiologist Lindblom, argue in favour of lumbago pain originating in the lower lumbar intervertebral discs. On puncture of the discs and injection of various solutions under pressure Lindblom was able to provoke pain similar to lumbago in the patient.

Another unsolved problem is whether a trauma can provoke a disc rupture. This point which is important from a practical standpoint has been much discussed but no clear answer has yet emerged.

The sciatic neuralgia syndrome caused by disc rupture has now been much better clarified and defined.

A distinction is made between subjective trouble and the objective findings by examinations.

Subjective Trouble. Sciatic pain is usually preceded by back ache. The period elapsing between backache and the true sciatic pain may vary considerably—it often extends however from a few days upto some weeks. The backache may appear as acute lumbago with severe aching at the slightest movement of the back.

True sciatic pain, a neuralgia os varying severity localised to the area embraced by the sciatic nerve may strike like lightning but may also develop and become more and intense over a period of days. The localisation of the pain is of the greatest value in deciding which lumbar root is being pressed. The usual localisation for the outlet of pain is the lateral part of the lower leg down towards the heel and the lateral toes, governed by pressure on S_1 root (the root which proceeds between the fifth vertebra and the sacrum). The next most common localisation is where pain radiates from the outer aspect of the thigh down towards the anterior aspect of the lower leg as far as the big toe. This arises from pressure on the fifth lumbar root. When the fourth lumbar root is affected pain does not usually pass down into the foot but remains on the anterior aspect of the thigh proceeding down over the front of the knee partly down the lower leg.

Pain on coughing and sneezing often occurs in disc rupture. It is presumably caused by pressure changes in the epidural venous plexus and in the cerebrospinal fluid combined with increased intra-abdominal pressure.

Paresthesis in the form of prickling and a creeping sensation down in the leg and also numbness often arises. The localisation of these discomforts can, like pain radiation, act as a guide to the nivesu diagnosis.

Paralyses. An affection of a nerve root can lead to a muscle paresis in the leg concerned which is at some time noticed by the patient as a slackness of the foot. Such a paresis must be of quite a high degree before the patient will perceive it but this does not in fact often happen.

Examination A sciatic patient attention must be paid to the following points :—The appearance of the back–the patient often stands with a straight lordosis or slight kyphosis of the lumbar spine together with a more or less advanced scoliosis (sciatic scoliosis). It seems to be most usual however that the patient leans the trunk over towards the affected side so that the scoliosis is thus convex to the healthy side. Tenderness to pressure above the spinal processes of the lower lumbar vertebrae may arise. If the patient then reports a pain radiating down the leg in question such information is valuable.

Lasegue's test. If the legs of the patient, who lies on his back will be bent at the hip with straight knees the patient feels intense pain in the affected leg. If the attack is severe it is not possible to left up the leg more than 10–20 before pain occurs. It is considered that pain in Lasegue's test originates when the nerve root affected is stretched over the disc prolapse. When the patient begins to be free of pain Lasegue's test becomes gradually negative. It may possibly be due to the nerve root adjusting itself to its position drawn tautly over the prolapse. Another explanation may well be that the disc protrusion shrinks and thus the tension in the nerve diminishes.

Palpation tenderness over the sciatic nerve is common,

Paresis. Paresis in the leg affected is a frequent sign, but careful examination must be made to discover it. This has appeared more and more in recent times. The most important practical point is whether there is weakness in the extensors of the toes, especially big toe paresis. Big toe paresis usually arises on compression of L_v root. On evaluating these paresis, frequently not of any severity, comparison must be made with the healthy side.

Muscle atrophy can sometimes be observed in the lower leg musculature. Occasionally it is quite striking at an early stage. It is important to record disturbances in sensibility in the lower leg.

The experience gained with the passage of time that an affection of the L_v root produces a reduction in sensibility on the anteromedial aspect of the lower part of the lower leg, forward over the ankle and out over the large toe now seems to be generally accepted. Pressure on the S_1 root reduces sensibility from the lateral part of the lower leg down over the lateral mellaeolus towards the lateral edge of the foot and from the heel out towards the little toe.

Reflex disturbances in sciatica have long been observed. Usually the achilles reflex disappears or is weakened in the affected leg. The disappearance of the achilles reflex argues in favour of a disc rupture between L_v and the sacrum, i.e. pressure on S_1 root. A disc rupture between L_{iii} - L_{iv} often demonstrates a weakened patellar reflex while pressure on L_v root does not produce any reflex disturbances.

Roentgen examination. Normal X-ray examination of the back is seldom of any help in localising a possible disc rupture. On the other hand a myelogram can give very clear data. In the Scandinavian countries positive contrast media (abrodil) are now chiefly used. In the clinic for which I am responsible in Malmo the myelogram has become routine procedure if a disc rupture is clinically suspected. The myelogram shows as impression in the contrast filled dural sac at the site of the disc rupture or a defect in the contrast filled sheath of the nerve root. A positive myelogram gives clear information about the localisation of the disc rupture and also gives a certain amount of data about its size.

Treatment. How then should a disc rupture with sciatica be treated? The question to be answered is generally—is the disc rupture to be treated conservatively, i.e. non-operatively, or should it be operated on?

The arguments is favour of conservative treatment are that it does not involve any obvious risk to the patient and also that one knows from experience that almost all sciatica patients become free from discomfort sooner or later. The argument against conservative treatment is found in the experience that sciatic pain is often very difficult to withstand and that even if it disappears it recurs easily and that generally it cannot be known how long it will last in any individual case. We are also aware from experience that many sciatica patients go about with varying degrees of severe invalidity without any possibility of resuming work for months, indeed years perhaps.

What methods do we employ for conservative treatment?

1. First of all bed rest, which means that pressure on the disc will be diminished, possibly with some kind of traction.
2. Immobilisation of the spine, usually in a plaster corset to avoid movement of the disc and also perhaps to distribute the pressure on the disc in a better way. Immobilisation of the disc in a plaster corset

is usually preceded by correction of the back's malposition. How far this correction should be taken is often a subject of lively discussion, some specialists prescribe almost violent corrections, others a slower correction under "traction". Undoubtedly such corrections often lead to the disappearance or lessening of sciatic pain but the disturbing part is that what is happening in the affected disc during the manipulations cannot be determined. Perhaps a more favourable distribution of the disc pressure is obtained, perhaps a disc-protrusion retreats, perhaps a rupture occurs in the protruded part of the disc so that it no longer exercises pressure on the nerve. More violent manipulations can cause permanent disturbances of nerve root function with paresis in the affected leg.

3. Heat treatment is a valuable accessory to conservative sciatica treatment. In Scandinavia it is often given in the form of short wave treatment.
4. Muscle massage of the frequently tensed lumbar musculature may also be valuable.
5. Physiotherapeutical treatment slowly carrying out an alteration in the carriage of the back, is seldom employed as long as true sciatic pain exists but is incorporated later when the discomfort consists more in backache or when it is important through improved carriage of the back to try and prevent the sciatica recurring.

In the operation treatment, by means of partial resection of the vartebrae archs on a level with the disc rupture, entrance is made through the ligamentum flavum; the dural sac and the nerve root, often stretched above the disc protrusion are held back and after incisions in the bulging disc the degenerated and loose disc tissue is extracted, often all of the nucleus pulposus. Previously a large resection of several vertabrae was performed but now this is very rarely necessary when a reliable niveau diagnosis is obtained prior to operation. Sometimes it is found at operation that a large part of the disc lies like a loose sequester around the nerve root.

The effect of a disc rupture operation is usually very striking. On the following day the patient is already free of the frequently severe sciatic pain and can often be allowed to sit up or walk one or two weeks after operation. Even if the sciatic pain completely disappears, the patient suffers after operation a certain amount of vague backache of the same type as conservatively treated patients after their sciatic pain has departed.

The argument in favour of operative treatment is that it directly attacks the disc protrusion pressing on the nerve. By virtue of this the sciatic pain disappears and the patient very soon becomes able to work and can resume normal life. With conservative treatment it is almost impossible to say how long the sciatic trouble will last.

The argument against operative treatment is that an operative intervention carries with it a certain immediate risk as with all operations. An infection may arise, a nerve root can be damaged, etc. With increased experience and better resources for combating post-operative infections or other general complications the risk in disc rupture operations must now be considered as very small.

Is then a patient operated on for disc rupture completely free in the future from sciatica? To this question the answer must be no. A new disc protrusion may arise from the previously affected disc and a disc rupture can occur in any of the adjoining discs. This may happen,

but we also know that sciatic recurrence from conservative treatment is much more common.

Our present attitude to the treatment can be stated as follows:—

In the Orthopaedic Clinic in Malmo we have worked out the following procedure. Operative indications are constant pain showing no tendency to diminish after 3–4 weeks treatment (bed rest, plaster corset.) This is further strengthened by serious paresis, i.e. much reduced peroneus function, but not by disturbances in sensibility or reflex trouble. The rapid onset of severe sciatica, making a total prolapse probable, may justify a shortening of the "waiting period."

If the patient has had previous attacks, successfully given conservative treatment, the same reasoning holds good. If the previous sciatic troubles have instead been particularly intense and lasting it often happens that the patient himself at an early stage of a renewed attack wishes to be operated on. The individual concerned has obviously been frequently informed by other "disc rupture" cases that an operation can bring freedom from pain more quickly than conservative treatment. Even if grounds do exist for testing the effect of conservative measures first of all, even in such a case there are often grounds at the same time which argue on behalf of shortening the waiting period, if no clear improvement is shown fairly quickly.

Discussion sometimes centres round the desirability or necessity of taking myelograms before operation. According to experience of our Clinic a myelography is particularly valuable since it almost always gives a correct guide to the localisation of the disc rupture. Therefore we hardly ever operate without previously taking myelograms.

The neurological niveau diagnosis is not as reliable as myelography. In individual cases the neurological symptoms may be completely lacking. In our experience however it is relatively rare that a sciatica case with severe pain does not betray atleast after the elapse of some weeks a form of neurological disturbance on thorough examination.

Sometimes neurological symptoms may be confirmed without myelogram changes. The protrusion is then usually localised so far laterally and caudally in the disc that the myelogram remains intact. Anaemias and the neurological condition however together make very often in such cases the required contributions to the neveau diagnosis.

DERMATITIS REPENS OF CROCKER

(CASE REPORTS)

by

Dr. T. V. VENKATESAN, M.B.B.S., F.C.C.P., F.D.S., *Madurai.*

THIS is an infectious dermatitis involving the extremities. The fingers are commonly affected than toes. The disease commences in the region of the nail and spreads slowly from this point. Trauma is a predisposing factor. The condition commences as deep seated vesicles with moderate inflammation. The infection leads to pustulation. The skin becomes oedematous and crusted. The overlying area becomes denuded leaving raw area with slight turbid fluid. The disease may extend to the palms from fingers and soles from toes.

Etiology :—The disease is said to be due to a particular strain of staphycococcus aureus and albus, Coagulase positive micrococci are also responsible.

Acrodermatitis continua Hallo peau is very difficult to distinguish from Dermatitis repens. The site of prediliction is similar. The general involvement of the various parts of the body is more common with Acrodermatitis continua of Hallapeau.

Histopathologically the two diseases can be distinguised namely : In dermatitis repens there is more marked pustulation with eczematoid changes. In Acrodermatitis continua the lesions are more psoriasiform namely there is acanthosis, papillamatosis and polymorphonuclear infiltration with migration of leukocyles through epidermis to form microabscesses.

The treatment of Dermatitis Repens.

The various measures found useful are:

1. Anti inflammatory treatment :
   (*a*) Hydrocortisone locally
   (*b*) Prednisolone orally.
2. Antiseptic treatment :
   Warm 1 in 4000 permanganate bath daily.
3. Antibiotic therapy especially synermycin capsules orally, or Erythromycin orally.

Two cases of Dermatitis Repens.

1. A. Lakshmi Ammal. Age 33.

Diagnosis : Dermatitis Repens of crocker.

Lab :—	W. B. C. :	12,400
	H. B. :	65%
	Skin scraping :	No fungus spores.
	Skin smear :	Secondary Organisms plus. No culture was done.

Clinical findings :—The patient came on 15—8—57 with a history of skin lesions, affecting the palms of 5½ months duration. The lesions are crusted in the fingers with interspersed raw areas with exudation. The palms show similar lesions. There is pruritis. The lesions commenced as vesicles which have ruptured.

Points in favour of Diagnosis :—

(*a*) The lesions commenced in fingers and gradually spread to palms.

(*b*) The fingers and palms are the commonest sites.

(*c*) Fungus infection is ruled out by the examination for spores.

Treatment given :— Local therapy :— The hands are immersed in warm 1 in

4000 potassium permanganate bath for 15 minutes daily. By mopping the area remove the crust gently. Then apply externally.

R/-	Silver Nitres	gr 10
	Brilliant Green	gr 10
	Gentian Violet	gr 10
	Methylated spirit	oz. 1
	Mix.	

Internal :—(*a*) Prednisolone tablets 30.

Dose :—1 tablet thrice daily for 5 days, twice daily for 5 days then once daily.

(*b*) Erythromycin (100 mgm) tablets 20.

Dose :—1 tablet thrice daily.

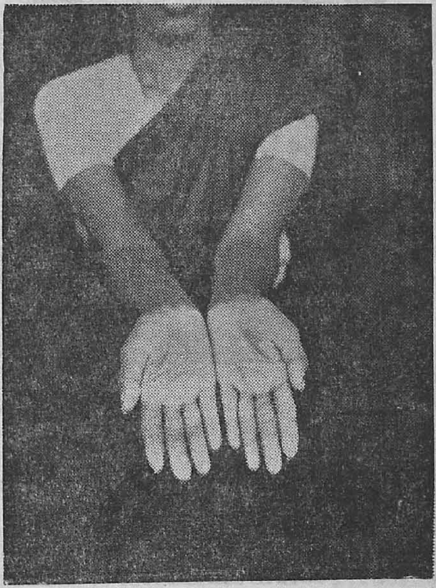
Injections :-(*a*) Sandosten with Calcium 5 cc × 8 amps.

1 amp I. V. twice weekly.

(*b*) Diamine Penicillin 6 lac × 12 vials.

Normal saline 5 cc × 12 amps.

1 vial in Normal saline I. M· twice weekly.



Progress :— Patient showed distinct improvement and the lesions regressed and cleared and the second picture was taken on 10—9—57.

* * *

2. Mr. C. S. Sundaram. Age 41.

Came to clinic in September 57 with vesicular, Pustular and crusted lesions over the fingers and raw areas over both palms showing exudations. Duration is about 3 months with history of itching.

Lab :–Skin scraping : No fungus spore· Secondary organism plus.

Blood for kahn : Negative.

No culture was done.

History :—The lesions commenced at the root of the nails on the dorsum of the fingers as small vesicles which spread to the palmar aspect of the fingers and palms.

Treatment given :—EF corlin Lotion. Externally after preliminary bath with warm 1 in 4000 potassium permanganate Lotion. He also received cortisone paranterally 25 mgm twice daily for 5 days and then once daily for 10 days. This was followed by Prednisolone 30 tablets in tailing of doses. Erythromycin was given to a total of 2000 mgm.

Progress :—The patient showed very good recovery. The second picture was not taken in this case.

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* PROBLEMS IN DIAGNOSIS WITH REFERENCE TO CARDIO-VASCULAR DISEASE

BY

Dr. P. K. KRISHNAN KUTTY, M.D. (Madras), M.R.C.P. (London), M.R.C.P. (Edin.), F.R.F.P. & S. (Glas.),

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THE introduction of the broad-spectrum antibiotics into therapeutics has served to control infectious diseases so thoroughly that degenerative vascular and neoplastic diseases have come to attract greater attention from medical practitioners in modern times. Therefore, it is of topical interest to discuss the problems arising with reference to cardio-vascular diseases. One is often asked to assess the cardio-vascular status of young individuals who are applying for jobs or people being examined for insurance purposes. What are the criteria for diagnosing normality of the heart ?

The apex best helps to serve as a guide to the size of the heart and if it is shifted outside the midclavicular line in the 5th intercostal space the heart is said to be enlarged. But it should be appreciated that scoliosis with a convexity to the right in the upper dorsal region will shift the apex beat to the left. So also does the pectum excavatum. In an obese individual, the diaphragm is likely to be raised and this may show a change in the position of the apex beat.

What is the significance of an apical systolic murmur ? Many a young man is turned down because of the presence of a systolic murmur at the apex. This is definitely not justifiable. Tachycardia, anaemia, fever and thyrotoxicosis can produce functional systolic murmurs. An organic systolic murmur is generally rough (grade III or above) either replaces or immediately follows the first sound and occupies the whole systole (i.e., pan-systolic). It does not change with posture or exercise. A functional systolic murmur is soft, does not replace the first sound and does not occupy the entire systole. It changes in character with change of posture, respiration and exercise. Persons with innocent murmurs should be considered fit and I think the rules for medical fitness should be modified accordingly.

The skiagram of the heart in an obese individual may show an apparent enlargement due to the presence of the pericardial fat at the apex, and the electrocardiogram may show a 'Q' wave in lead III which is not pathological.

Many obese patients have difficulty in breathing which is made worse on exertion and on climbing of stairs. Some of them have oedema of the lower extremities as well. So these cases are mistaken for congestive cardiac failure. But careful examination will show that their lung bases are clear and their liver non-tender. These patients do not require digitalis but they should be put on 1000 calories diet to which type of treatment they respond remarkably well.

In clinical practice, many of us would have come across cases of paroxysmal dyspnoea coming on at night. Some times there is a real difficulty to differentiate an

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* Read on the occasion of the Annual Meeting of the District Medical Association at Chingleput in October 1957.

attack of bronchial asthma occurring in a patient with hypertension from hypertensive cardiac failure. Bronchial asthma is a chronic disease and so the patient's history is generally of many years duration whereas cardiac asthma is usually an acute medical emergency. In cardiac asthma pre-systolic gallop rhythm is common. The circulation time is normal in bronchial asthma whereas it is prolonged in cardiac asthma.

Praecordial pain is a very common symptom which brings the patient to the doctor. It is extremely important to separate true cardiac pain from non-cardiac pain. In true cardiac pain, the pain is generally retrosternal but some times it may be in the abdomen. Abdominal pain is of some significance. I have seen many cases of myocardial infarction where the pain had been abdominal and which has been mistakenly diagnosed as flatulence and dyspepsia. I had one patient who complained of severe pain in his teeth and its significance is obvious. Cardiac pain is generally constricting in nature. Some patients say that they have a chocking sensation. Recently I had 5 cases who complained of a burning sensation in the chest and they had typical evidence of acute myocardial ischaemia, proved by electrocardiogram. A few years ago one used to be dogmatic about the nature of the pain in myocardiac ischaemia. If it is not constricting or choking in nature, serious myocardial disease used to be excluded. But recent experience has shown that the nature of pain may vary from case to case with coronary arterial disease.

In angina of effort the pain is the same as in coronary thrombosis, but it comes on with exertion and is relieved by rest or vasodilators. Many patients get symptoms when they exert on a full stomach. The pain is often arresting in nature and the patient feels that he may die if he does not stop all activities immediately. The fact that the pain comes on when the stomach is full may give an impression that the offending organ is the stomach. When in doubt comparison of the electrocardiogram taken before and after exercise may be helpful. The electro-cardiogram is usually normal before exercise, whereas it may show depression of the S. T. segments immediately after exercise. This finding is diagnostic.

Now-a-days, the diagnosis of coronary thrombosis is being misused to label all causes of sudden death. One of the most important diseases which is often misdiagnosed as coronary thrombosis is massive pulmonary embolism. Usually massive pulmonary embolism occurs in patients who are confined to bed whereas coronary thrombosis occurs in people who are active and moving about. The pain in pulmonary embolism is maximum from the outset whereas in coronary thrombosis the pain increases gradually in intensity to reach a maximum in the course of a few hours. Cyanosis and dyspnoea are commoner in pulmonary embolism. Generally the source of embolism can be detected as phlebothrombosis in the lower extremities and by a positive Homan's sign. X-ray of the chest shows increased translucence in pulmonary embolism whereas in coronary thrombosis congestion of lungs is commoner. The E. C. G. may be normal, may show right bundle branch block or some times may show a 'Q' wave in III in pulmonary embolism, AVF being normal.

Palpitation is complained of by many patients. The condition may be due to extra-systoles in which case, the patient requires only re-assurance. Some times it may be due to paroxysmal auricular tachycardia. This is characterised by a sudden rise in heart rate reaching 160 and some times more. The patient complains of palpitation, fainting or a feeling of fluttering over the chest. Only rarely does the doctor have the occasion of seeing a patient in an actual attack. A patient aged sixty developed paroxysmal

tachycardia at 4-30 A.M. in the morning, one day. He was in great distress and his family physician was struggling with the patient to control the pulse rate till 6 A.M. at which hour I was summoned to the patient's bed side. With a gentle pressure on the right carotid sinus and with simultaneous ausculation of the heart, the heart rate settled down to normal in the course of a few seconds. Obviously the family physician had not been able to locate the patient's carotid sinus and apply pressure.

On certain occasions one comes across cases of cardiac failure in which the aetiology is not quite clear. Under those circumstances it is advisable to remember the extrinsic factors which may precipitate cardiac failure, like anaemia, beri beri, corpulmonale, Paget's disease of the bone and thyrotoxicosis.

Hypertension is a very common disease and the diagnosis is incomplete without investigating its aetiology It is always easy to diagnose essential hypertension since its aetiology is uncertain. It is safe to diagnose secondary hypertension in people below the age of 50. The minimum investigations in the study of hypertension should include examination of eye grounds, renal function and an intravenous pyelography and estimation of 17 keto steroids and nor-adrenaline in urine. In all young persons with hypertension it is worth the while to take the blood pressure in the lower extremities. Hypertension in upper extremities and hypotension in the lower extremities is often characteristic of coarctation of the aorta.

In the above lines I have attempted to indicate the difficulties in diagnosis which occur in the course of clinical practice and ways and means by which a clinician can arrive at the correct diagnosis.

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* DIAGNOSIS AND TREATMENT OF SMALLPOX

by

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SMALL-POX is caused by a filterable virus identical with that of vaccinia and is characterised by an initial stage of toxaemia, followed by a focal eruption which appears in a definite regional order and attains centrifugal distribution. The focal lesions at first papular, become successively vesicular and pustular. Typically, pustulation is accompanied by a secondary illness due to septic absorption.

Small-pox in epidemic form, is common in India and at present there is an epidemic in Erode in our district and also the neighbouring districts of Salem and Trichy. So it is essential that the disease has to be correctly and quickly diagnosed from the other common eruptive fevers like chickenpox, measles, Herpes and skin diseases :

(i) Children : papular urticaria ;

(ii) Adolescents : pustular acne.

(iii) Adults : pustular syphilides ;

(iv) Erythema multiforme exudativum

(v) Drug Rashes : Sulphonamides, iodides, bromides and copaiba.

For the correct diagnosis it is essential that the examination of patient must be done in a systematic manner. It is essential that the light should be good and daylight is far preferable to artificial light. The clothes should be removed as far as possible and the patient wrapped in a blanket, placed upon a couch or bed. The examiner first inspects the state of vaccination and this in itself may be sufficient to exclude smallbox - A history and evidence in the form of foveated scars of recent vaccination may exclude a diagnosis of smallbox. Possibility of vaccination having been performed upon unusual sites-in the thighs in some western ladies-should always be borne in mind. Next the fauces and buccal cavity are examined for the presence of lesion. The blanket is than opened out and the patient directed to fold the arms across the chest. Then the examiner standing at the foot of the bed, observes the general and relative distribution of the eruption. Particular attention is paid to the gradations of density of the lesion upon the face, arms, trunk and legs : to the arrangement of the lesions in a centrifugal, centripetal or haphazard manner and to that profusion or otherwise upon prominences or in hollows respectively. Only when these features have been determined should attention be paid to the character of individual lesion.

The usual incubation period to the first appearance of smallpox rash is fourteen days. The clinical stages of an attack of smallpox on (i) the prodromal, pre-eruptive toxaemia (primary viraemia) followed by (ii) the appearance and evolution of the focal eruption (secondary viraemia) which passes through various stages.

In unmodified smallpox, the onset of the toxaemia of smallpox is usually abrupt. Headache, backache, vomiting and pyrexia form a common syndrome of which the headache usually frontal and accompanied by giddiness, and pyrexia (103°or104° F) with proportionate tachycardia are constant features ; pain in the limbs are less common. Shivering is common, rigors in adults or convulsions in children are occasional events. Cough and bronchitis are not unusual, nor is sore throat which is strictly not a toxaemic

* Reprinted from the Coimbatore Dist. Medical Association Annual Number (1956)

symptom but connected with appearance of exanthem upon the plate and fauces. Prostration is considerable. The face has an "ironed-out" appearance-characteristic greyish fatigued expression due to less of tone of the small muscles. Conjunctivae may be suffused. Tongue is furred, mouth dry and breath offensive. Although physically prostrated, patients may be mentally alert, too alert since insomnia is common. This stage lasts for two or two and a half days but periods of three days are not uncommon. During the Prodromal stage, in some cases prodromal rashes occur. They are (i) haemorrhagic in toxic or haemorrhagic smallpox and indicative of a severe or fatal attack. (ii) erythematous in mild attacks and (iii) urticarial.

In typical cases in the earliest stages the rashes are *macular* and not raised above the surface of the skin. They are of pin head size. In a few hours the macules become definitely papular. The papules are deepset in the skin, dark pink in colour 1/8″ or more in diameter and shotty when rolled between finger and thumb. Surrounding each lesion is an areola of lighter pink and of variable width. Papules continue to appear until the eruption of the papular stage is complete. This process occupies two days sometimes three days. Then the conversion of papules into vesicles commences with these lesions which were the earliest to appear. Breaking down commences at the top and in 24 hours become clear vesicles. After 24 hours they become turbid. The early *vesicle* is plump and well filled, the apex is rounded but sometimes flat. It is circular not elliptical, diameter being 1/4″ or less. During the later stages the vesicle show phenomenon known as "Umbilication"– a dimpling of the apex due to less of fluid tesion. Vesicles are multilocular. The areola increases in width and attains its maximum during the first day of the vesicular stage. Vesicular stage lasts about two days and then merges into the *pustular* stage which lasts for four days. During the latter two days of this stage pustules enlarge in size and may reach a diameter of 3/8 of an inch, when they attain the stage of maturation. By this time areola mostly disappear.

During the next 8 or nine days pustules gradually dry up forming ultimately dark brown almost black scabs or "seeds" which take a number of days to separate. On the palms and soles they take a large number of days before separation is complete. Circular scars are left: the bases are at first pigmented but they ultimately become avascular cicatrices or pocks.

In smallpox–Exanthem - earliest focal lesions appear upon the buccal, faucial, pharyngeal and bronchial mucosa. Owing propably to the delicate nature of the structures on which they occur, their evolution is more rapid than the cutaneous lesions, and are seen to be in a later stage. Since the existence of lesions in mouth is an important point in the differential diagnosis of doubtful cases their presence should always be ascertained.

Exanthem : Order of appearance and density :

Face, then arms, next trunk particularly back and lastly legs. Density of lesions is always greater on the face and arms. They show a centrifugal distribution, that is-although every anatomical region of the body is invaded in the order and density mentioned above when any region is examined separately it is seen that the distant parts bear a relatively greater profusion of lesions than those which are proximal. On the trunk lesions are scanty or relatively scanty; fore-arms and hands are more affected than upper arms; legs and feet are more affected than thighs. Lesions are more numerous upon the *extensor* surfaces than the *flexor* surfaces of limbs. There is a tendency

for lesions to occur upon convexties and prominences-brow and prominences of malar bones: bridge of nose, point of chin, pinna, prominence of sternomastoid clavicle back of the neck and the scapula. It occurs in the area of pressure, irritation or inflammation also, i. e. pressure lines of braces or garters occupational pressure points on the fingers, scratches or sunburns are examples.

Unmodified small-pox is classified as :

(1) Toxic or haemorrhagic

(2) Confluent

(3) Discrete.

Toxic variety is invariably fatal and the patient dies before the appearance of focal / eruptions.

Differential diagnosis :

1. *Chickenpox* (*a*) prodromal illness is lacking or trifling.

   (*b*) Centripetal distribution of the eruption.

   (*c*) Relative profusion of lesions in concavities-for example axilla and orbit.

   (*d*) lesion in all stages.

   (*e*) Superficiality of lesions.

   (*f*) occurrence of elliptical lesions.

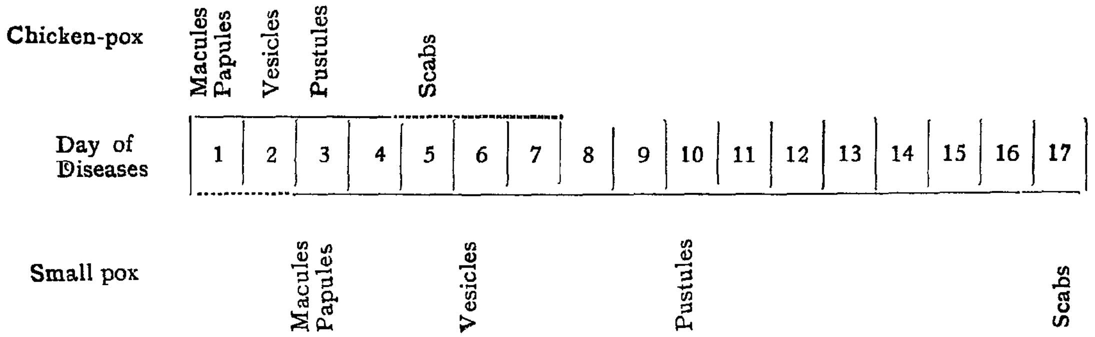

Diagram comparing the Evolution of the Focal Lesions of Chickenpox and Smallpox, Note the more leisurely development of the lesions of Smallpox.

Measles : In the early stages velvety maculopapules on the forehead in severe cases of measles may be mistaken for similar lesions in small-pox, but the presence of Kopliks spots and the subsequent evolution of the eruption are conclusive of measles.

Herpes : Generalised infection of herpes may resemble mild small-pox but laboratory test helps to differentiate between the two.

Skin-diseases :

(1) *Children* : Papular urticaria-lesions absent or sparse on the face. Never present in the mouth. Abundant around ankles. The lesions are small superficial papules. Some times wheals may be found. There are usually scratch marks and frequently evidence of old lesions, and the child is in good health.

(2) *Adolescents* : Pustular acne distribution is on the face, back and upper chest. Skin is greasy with comedones (black heads) and usually abundant scarring as a result of chronicity.

(3) *Adults :* Pustular syphilides : The distribution is not centrifugal. Usually pustular from commencement but some times some pauples may be seen mixed with pustules. Other signs of syphilis

and W. R. serve to distinguish. Note the possibility of concurrent small-pox and syphilis.

Erythema multiforme exudativum. Here large superficial bullae appear. Particularly on the backs of the hands and upon the legs, less abundantly on the face. Lesions occur in the mouth. Panophthalmitis is especially prone to occur.

Drug Rashes : Sulphanimides, Iodides, bromides and copaiba. There is a history of administration of the particular drug and the distribution of the rashes does not conform. The typical Sulphonamide rash is morbilliform.

Drug rashes will disappear on discontinuing the administration of the drug.

Complications :—

(1) *Cardiac* : Myocardial failure may occur during the stage of toxaemia in the toxic or haemorrhagic forms and during secondary illness which accompanies maturation of the pustules.

(2) *Nervous* : Apart from delirium or convulsions nervous symptoms varying from extreme drowsiness to complete loss of consciousness, may occur at the commencement of illness and disappear completely at the end of toxaemic phase. Peripheral neuritis and psychoses occasionally occur.

Respiratory :—

Some degree of bronchities with or without laryngitis is usual in the more severe attacks. Broncho-pneumonia and less commonly lobar pneumonia may be terminal and fatal events.

Eyes : Simple conjunctivitis is common. They may become secondarily infected with resulting keratitis and destruction of the globe. In this case, excision of the affected globe is necessary to prevent occurrence of sympathetic ophthalmia.

Ears : Otitis media is an occasional extraneous complication.

Genito-urinary : The pregnant woman may abort at any stage of an attack and disturbances of menstrual function are not uncommon. Orchitis is an occasional complication.

Skin : Pustular dermatitis and boils may be troublesome in convalescence. Occasionally gangrene of the skin occurs usually on the distal parts of the limbs.

Treatment of small pox :—Consists largely of skilled nursing. As much rest as possible is essential because of the effects of the disease upon the myocardium. At the same time regard to posture is most important because of the liability to pulmonary complications and to pressure sores. A mattress of the "sorbo" rubber type is preferable. Continuous attention to the hygiene of the mouth, eyes and skin is essential. Owing to the liability to delirium of violent type and the tendency of the small-pox patient, in his delirium to get up from bed and run about or even run away sufficient assistance must always be available to imposs the necessary restraint.

The diet must be as liberal. In confluent small-pox in the stage of maturation. nasal feeding may be necessary owing to the swollon condition of the face.

The head-ache and pains in the back and limbs usually respond to drugs such as aspirin but my necessitate an injection of morphine. Delirium may be controlled by chloral and bromides or paraldehyde. The condition of the heart may be ascertained frequently and cardiac drugs such us nikethamide prescribed if necessary.

In the early stages, administration of sulphanomides and/or penicillin given as prophylatic against the secondary infection (not the virus), is useful.

Repeated bathing of the pustules with an alkaline lotion containing some deodorant is comforting to the patient and his attendants alike.

Separation of the scabs must not be effected by force, for the traumatic lesion which results will take a longer time to heal than that occupied by the separation of the scab in the ordinary way.

Convalescence from smallpox may be protracted and after release from isolation a prolonged period of rest, fresh air and tonic treatment are desirable.

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*URINARY TRACT INFECTIONS AND THE NEW ORAL NITROFURAN "FURADANTIN"

by

Dr. W. R. BETT, M.R.C.S., L.R.C.P., F.C.S.,

Medical Consultant to Messrs. Menley & James Ltd., & Smith Kline & French Laboratories Ltd., London.

THOUGH Sulphonamides and Antibiotics have been very useful in the treatment of many infections, certain bacterial strains are resistant to these drugs, and hence the need for new antibacterial drugs had become necessary, particularly for infections of the urinary tract. In the year 1944 the preparation of Nitrofurazone 'Furacin' was produced, and this drug was found to be dramatically successful in the topical treatment of chronically infected wounds and burns. Further experimentation with nitrofurans has produced Nitrofurantoin N–(5-nitro-2-furfurylidene)–1–aminohydantoin, one of a series of substitued furan derivatives having a nitro group at the 5-position in the furan ring. Nitrofurantoin, known by its trade name 'Furadantin', was introduced in 1951 by Messrs. Eaton Laboratories Inc., Norwich, N. Y. It is unrelated to the Sulphonamides or the Antibiotics. 'Furadantin' has been accepted in the United States for inclusion in the N. N. R., and it is category I drug in the U. K.

Bacteriology:—'Furadantin' acts by interfering with the enzymatic metabolism of the bacterial cell. It possesses a broad antibacterial spectrum that encompasses both gram-positive and gram-negative organisms, especially those commonly found in urinary–tract infections. It does not inhibit fungi or viruses. The drug remains effective in the presence of serum and of pus.

Pharmacology:—'Furadantin' acts on the urinary tract. Given by mouth, it appears in the urine within half an hour; 45 per cent of the oral dose is excreted in the urine in eight hours. The turbid urine often clears within 24 hours and in 3–5 days the urine is free of pus cells. Within one week the urine usually becomes sterile. The drug is highly soluble in urine as much as 0·2 mg./ml. at a pH of 7·5; thus there is no risk of precipitation of 'Furadantin' crystals in the urinary tract. The use of 'Furadantin' does not hinder future systemic medication.

The average adult dose is eight 50 mg. tablets a day, 2 tablets with each meal and 2 at bed time. When more rigid control of dosage is required, the range suggested is 5-8 mg. per kg. body weight in a 24 hour period: a quarter of this dose being administered four times daily with food. If the responsible organism proves refractory, the dose may be increased to a maximum of 10 mg. per kg. (4·5 mg/lb). If this should produce severe nausea, reduce the dose, but not below 5 mg./kg. (2·2 mg./lb) in 24 hours. The average dosage should be maintained constantly for 5–7 days at least and for 3 days after the urine has been sterile. but for not more than 14 days altogether. If definite improvement is not seen within 7 days, the drug should be discontinued, The course of treatment may be repeated, if there is a recurrence, after a rest period of 4 weeks.

Furadantin, because of its rapid action, is a most useful drug in acute infections of the urinary tract and is effective against organisms resistant to the Sulphonamides

* Summary of a lecture delivered at the Madras City Branch on 30—4—'57.

and the Antibiotics, such as Proteus and Pyocyaneus. Typical indications are cystitis, prostatitis, pylonephritis, and pyelitis of pregnancy. It has no serious side effects and is safe to use in children and in pregnant women.

A large number of papers and clinical reports have been published on the therapeutic uses of 'Furadantin'. A few of the reports mentioned are (1) In a recent number of *The Practitioner* E. J. Wayne, Regius Professor of the Practice of Medicine, University of Glasgow, discussing urinary–tract infections writes: In the palliative treatment of chronic infections where the cause cannot be removed B. Proteus (Proteus vulgaris) is a commonly isolated organism. Such infections are best treated by 'Furadantin'. (2) In the British Medical Journal of August 4, 1956, Mary G. McGeown, M.D., Ph.D. of the Department of Medicine, Queen's University, Belfast has reported on treatments carried out with 'Furadantin' on 21 cases of refractory urinary infections associated with the presence of renal calculi or persisting after the removal of calculi. In 48% of cases the urine became sterile after 14 days and in 28% the urine remained sterile for three months. Dr. McGeown has reported that in cases of refractory urinary infections it would appear that 'Furadantin' is a drug of considerable value. (3) Of the many articles published in American Journals an important one is the report by G. Carroll and R. V. Brennan in 1954. They have confirmed that in a series of 128 patients, 'Furadantin' was particularly useful in the stubborn Proteus infections. 21 out of 25 patients with Proteus were controlled by 'Furadantin' treatment. (4) In the A. M. A. Journal of Diseases of Children of Feb. 1955 S. H. Johnson and M. Marshall have reported after having treated 38 children that 'Furadantin' is an effective relatively nontoxic urinary antibacterial agent in paediatric practice. (5) E. S. Diggs and colleagues have also reported in 1956 that 'Furadantin' was used to treat acute and chronic urinary tract infections in 11 pregnant women and successful results were obtained.

'Furadantin' is a useful oral, specific agent for the treatment especially of acute urinary-tract infections and is the best drug available today for treatment of Proteus Vulgaris. Its side effects are very few and consist mostly of nausea and vomiting, which were best minimised by taking each dose with a meal and the last dose before retiring with warm milk or a light snack. 'Furadantin' will help the Urologist, Gynaecologist, Obstetrician, the Paediatrician and General Medical Practitioner in quick and efficient eradication of the various infections of the urinary tract.